



Nephrology Times

Practical News, Trends, and Analysis

July/August 2026

VOLUME 18, NUMBER 6

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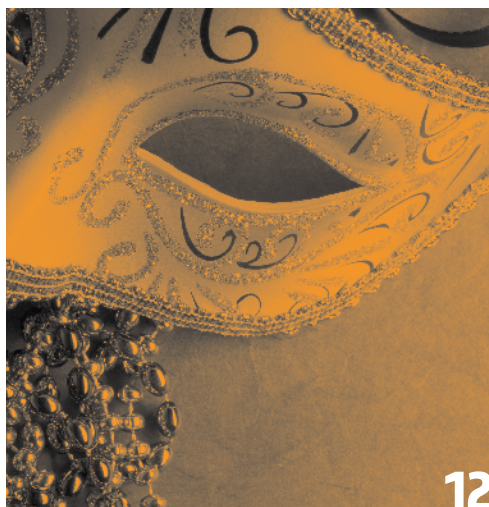
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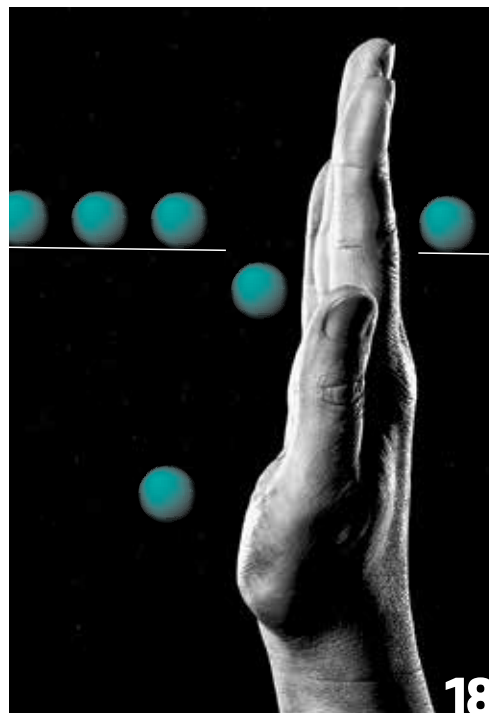
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Turning Risk Into Action

How nephrology registered nurses and advanced practice registered nurses drive value-based kidney care



Molly Cahill
MSN, APRN-BC, ANP-C, CNN

By *Molly Cahill, MSN, APRN-BC, ANP-C, CNN*

CKD affects more than 37 million adults in the United States and represents one of the most expensive chronic conditions covered by Medicare. According to 2025 United States Renal Data System data, once an individual experiences progression to kidney failure requiring dialysis, the average annual cost for treatment is \$94,000. Yet the greatest opportunity to affect the outcomes and costs occurs years earlier—during routine primary care where clinical decisions determine whether CKD progression accelerates or stabilizes.¹

Value-based kidney care models are increasingly designed to identify risk earlier, coordinate care across settings, and prevent avoidable disease progression. But predictive tools and clinical guidelines alone do not change outcomes. Their impact depends on the clinicians who translate data into action. Across nephrology practices, that role is often filled by nephrology registered nurses (RNs) and advanced practice registered nurses (APRNs), whose work in monitoring risk, coordinating care, and engaging patients shapes the trajectory of kidney disease long before dialysis is required.

The Gap Between Guidelines and Real-World CKD Care

Over the past decade, the clinical toolkit for slowing CKD progression has expanded significantly. Evidence-based interventions such as BP control, renin-angiotensin system blockade, sodium-glucose cotransporter 2 inhibitors (SGLT2i), and regular monitoring of albuminuria are known to reduce the risk for kidney failure and cardiovascular complications and are emphasized in current clinical practice guidelines for CKD management.²

Despite these advances, implementation in real-world practice remains inconsistent. Albuminuria testing is often underutilized, BP targets are not consistently achieved, and initiation of kidney-protective medications may be delayed. In many cases, patients are referred to a nephrology professional only after substantial loss of kidney function has occurred.

These gaps rarely result from one missed intervention; they accumulate gradually over months and years of care. A lab test is delayed. Medication titration is postponed. Follow-ups are missed. Over time, these issues grow, allowing CKD progression to accelerate silently. Value-based kidney care models aim to close the gaps by shifting from episodic treatment to proactive disease management. However, doing so requires clinicians who can continually monitor risk, identify care gaps, and intervene before complications occur.

The Workforce Translating Risk Into Action

Within nephrology practices, RNs and APRNs frequently serve as the operational core of this proactive care model. Their roles extend beyond traditional clinical encounters to include risk monitoring, care coordination, and patient engagement across the CKD continuum.

One of their most important functions is the early identification of risk signals. Changes in estimated glomerular filtration rate, a rising urine albumin-to-creatinine ratio, or worsening BP control may indicate accelerating kidney injury before symptoms appear. By recognizing these trends early, clinicians can initiate interventions that stabilize kidney function and prevent acute deterioration.

Medication management also plays a critical role. Ensuring appropriate initiation and titration of therapies such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and SGLT2i requires ongoing monitoring, patient counseling, and follow-up. Nephrology RNs and APRNs often lead these efforts, working with physicians to optimize treatment plans while addressing barriers such as medication tolerance, affordability, and adherence.³

Equally important is the role of patient education. CKD management depends heavily on patient engagement in areas such as BP monitoring, medication adherence, diet, and recognition of early symptoms. Through counseling and ongoing communication, nephrology RNs and APRNs help patients understand the significance of laboratory trends and treatment goals, empowering them to participate actively in their care.

Finally, care coordination remains a central responsibility. Individuals with CKD frequently interact with multiple healthcare professionals, including primary care clinicians, cardiologists, endocrinologists, and vascular specialists. Coordinating laboratory testing, referrals, and treatment plans across these settings reduces fragmentation and ensures that guideline-based care occurs consistently.

Why This Matters in Value-Based Kidney Care

The growing emphasis on value-based care reflects a broader shift in healthcare policy: improving outcomes while controlling costs. For CKD, this means preventing avoidable hospitalizations, delaying CKD progression, and maintaining individual quality of life.

When evidence-based interventions are implemented early and consistently, the impact can be substantial. Improved BP control slows kidney function decline. Early treatment of albuminuria not only delays CKD progression but also reduces cardiovascular risk. Timely nephrology involvement allows management of CKD complications and preparation for kidney replacement therapy, avoiding emergency dialysis initiation.

These clinical improvements also translate into economic value. Preventing hospitalizations, reducing emergency department visits, and delaying dialysis initiation all lower the total cost of care. Importantly, these gains are not typically achieved through a single breakthrough therapy but through the consistent application of multiple smaller interventions over time.

The clinicians who ensure that these interventions occur reliably are therefore central to the success of value-based kidney care models.

Predictive Tools Need Clinical Operators

Advances in predictive analytics are increasingly being integrated into kidney care programs. Algorithms can analyze laboratory trends, comorbid conditions, and utilization patterns to identify those at increased risk for CKD progression or hospitalization.

However, data alone do not change outcomes.

Predictive tools can highlight increasing risk, but clinicians must determine how and when to intervene. Without the clinical workforce to respond to these signals, predictive systems risk becoming passive dashboards rather than drivers of improved care.

Nephrology RNs and APRNs serve as the bridge between predictive insights and patient care. By reviewing laboratory trends, conducting patient outreach, coordinating follow-up testing, and initiating treatment adjustments, they transform risk data into actionable care plans.

In this way, predictive analytics and clinical expertise operate together. Technology identifies patterns of risk, and clinicians apply contextual knowledge and patient-centered judgment to determine the most appropriate response.

Looking Ahead

As value-based kidney care models continue to evolve, their success will depend not only on new therapeutics or predictive technologies but also on the clinical workforce capable of operationalizing the advances. Nephrology RNs and APRNs provide clinical expertise, patient engagement, and care coordination in ways that directly influence the trajectory of CKD.

The most meaningful opportunities to improve kidney health occur long before dialysis begins. By identifying risk early, closing care gaps, and ensuring consistent application of evidence-based therapies, nephrology RNs and APRNs help shift kidney care upstream—improving outcomes for patients while delivering the value that current healthcare systems increasingly demand. ●

Molly Cahill, MSN, APRN-BC, ANP-C, CNN, is a nephrology nurse practitioner in value-based care and president-elect of the American Nephrology Nurses Association.

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What Sparsentan's Approval Means for FSGS Care

By Charlotte Robinson

The nephrology community received welcome news on April 13, when the FDA approved sparsentan for the treatment of focal segmental glomerulosclerosis (FSGS) in patients aged 8 years and older without nephrotic syn-



Kirk Campbell, MD

drome. Sparsentan, a dual endothelin and angiotensin receptor antagonist previously approved to treat IgA nephropathy, is the first and only drug approved for FSGS therapy. The approval was supported by data from the phase 3 DUPLEX study, which showed that treatment with sparsentan resulted in a 46% reduction in proteinuria at week 108 compared with a 30% reduction with irbesartan.

We spoke with **Kirk Campbell, MD**, a professor and the Renal Division chief at the University of Pennsylvania Perelman School of Medicine and president of the National Kidney Foundation, about the importance of the approval for clinicians and their patients.

This transcript has been edited for clarity.

Before sparsentan's approval, how were you managing patients with FSGS without nephrotic syndrome—and where were the biggest gaps?

Before the approval of sparsentan, we were using primarily off-label medications to manage FSGS. While utilizing foundational therapy—typically ACE [angiotensin-converting enzyme] inhibitors, angiotensin receptor blockers—we would consider patients being eligible for immunosuppressive agents like systemic corticosteroids or calcineurin inhibitors versus other immunosuppressive agents, depending on the etiology [that] we thought FSGS was from, primarily, that would guide our decisions on which treatment approach to undertake. The options were limited to agents not developed specifically for the treatment of FSGS, and [we had] nothing with the detailed mechanistic rationale for utilization in patients that have this condition.

With sparsentan's approval, what will change for you in the clinic when you're seeing one of these patients?

It's great to have an option that's FDA approved, meaning that we've met the efficacy and safety thresholds, and we understand the mechanistic rationale for endothelin-1 causing podocyte injury and various downstream and secondary effects as well on the tubulointerstitial compartment, vasculature, etc. Having that mechanistic rationale—the biological plausibility

of an agent developed specifically to treat FSGS—means that we can speak more confidently to patients about why we're choosing this agent and why it may be effective for reducing the level of proteinuria that they have.

The DUPLEX trial showed substantial and durable proteinuria reductions, with more modest differences in estimated glomerular filtration rate (GFR) slope over 2 years. How should nephrologists interpret these findings when assessing long-term kidney protection in FSGS?

There's a strong biological plausibility and link between proteinuria reduction and stabilizing podocytes from injury—mountains of preclinical data and a lot of evidence really pointing to proteinuria reduction as a key clinical and therapeutic goal in managing patients with FSGS. We know from some of the preliminary evidence from the PARASOL project, for example, that the GFR slope metric that may be applicable in several other kidney disorders does not work so well in defining efficacy and how we should really consider success when we're managing patients with FSGS.

We've learned a lot over the years to value—at least for some of the short-term metrics—proteinuria reduction a little bit more than GFR slope, specifically for FSGS.

It's very noisy in FSGS. We see a lot of patients with hyperfiltration initially, with GFR declines later, [which is] especially problematic in children. We've learned a lot over the years to value—at least for some of the short-term metrics—proteinuria reduction a little bit more than GFR slope, specifically for FSGS. Now, in the long run, we ultimately do want to keep patients off dialysis. Long-term kidney function protection is what we're really striving for. But we do rely a lot more now, based on evidence, on proteinuria reduction as a key end point.

Which patients are you thinking about first for this drug, and how early in the course of FSGS do you see it fitting?

Whenever there's a new drug approval, we try to read the label very carefully. We look at the study population in the

phase 3 study—in this case DUPLEX—as well as earlier studies to align as best we can around the population being studied and who we’re seeing in our office just to have that patient-centered communication.

We’re well aware that individuals without nephrotic syndrome are really the group that this was approved for with FSGS diagnosis. That’s a large bucket. A lot of patients fit those categories. Those are individuals that have primary genetic secondary etiologies with the histologic lesion of FSGS being present. As part of a comprehensive strategy to reduce proteinuria, we have, certainly, a broad group of patients that we can have this patient-centered communication with to define when it would be most appropriate.

When you sit down with a patient who’s been through years of off-label therapy, how does this approval change the conversation?

We can certainly speak confidently that here’s a drug that was approved for FSGS. It’s the first time we’ve ever been able to

say that. The other conversations made it seem that we weren’t speaking too confidently about the rationale for prescribing a particular agent. We know that there are patients who are going to require additional therapy, together with approaches like sparsentan, but at least we can confidently say, “This drug has been demonstrated to reduce proteinuria in patients like yourself.”

Do you have any other thoughts to share with fellow clinicians?

I’d say this is the beginning of, hopefully, a lot of innovation in this space. A number of advocacy organizations, and certainly sponsors, have been quite active, and, hopefully, we can continue to innovate and do more for patients with conditions like FSGS in the years to come. ●

Scan to watch a video of our interview with Dr. Campbell.



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Conference Coverage

New Orleans, Louisiana | May 6-10, 2026

NATIONAL KIDNEY FOUNDATION SPRING CLINICAL MEETINGS

Nephrologists, fellows, and residents with a special interest in kidney disease, general internists, pharmacists, physician assistants, nurse practitioners and nurses, technicians, social workers, and renal and clinical dietitians attended the 2026 National Kidney Foundation Spring Clinical Meetings in New Orleans, Louisiana, to learn about developments in all areas of nephrology practice and network with colleagues. Presenters reported the latest insights into chronic kidney disease care, and participants were informed about new and evolving concepts related to kidney disease.

IMAGE CREDIT: ISTOCK.COM/DNY59

Iptacopan Shows Sustained Benefits for Treating IgAN

In **IgA nephropathy (IgAN)**, glomerular inflammation and kidney function decline are driven by alternative complement pathway overactivation. In APPLAUSE-IgAN, a double-blind phase 3 trial, researchers evaluated iptacopan, a complement factor B inhibitor, in adults with IgAN. All patients had a 24-hour urine protein-to-creatinine ratio (UPCR) of 1 g/g or greater despite optimized supportive care and an estimated glomerular filtration rate (eGFR) of 30 mL/min/1.73 m²/year or greater.

Patients were randomly assigned 1:1 to receive iptacopan 200 mg (n=238) or a placebo (n=239) for 24 months. At 9 months, results showed that iptacopan reduced 24-hour UPCR by 38.3% compared with placebo, with 43.9% of patients receiving iptacopan achieving 24-hour UPCR less than 1 g/g versus 17.5% of those receiving placebo.

Twenty-four month results presented by **Dana Rizk, MD**, and colleagues, showed that iptacopan significantly slowed eGFR decline over the treatment period compared with placebo, with a treatment difference of +3.02 mL/min/1.73 m²/year. The 24-hour UPCR reduction from baseline was greater in the group receiving iptacopan across all subgroups. A composite kidney failure end point (sustained ≥30% eGFR decline vs baseline, sustained eGFR <15 mL/min/1.73 m²/year, maintenance dialysis, kidney transplant, or death from kidney failure) was reached by 21.4% of patients receiving iptacopan and 33.5% of those in the placebo group.

In addition, fewer patients receiving iptacopan experienced hematuria compared with those receiving placebo at 24 months. Treatment-emergent adverse events (TEAEs) occurred at similar rates in both groups, and most were mild or moderate, with no deaths.

Overall, these findings indicate that iptacopan is associated with sustained kidney-protective effects, including slower functional decline, reduced proteinuria across subgroups, and decreased hematuria, compared with placebo. Combined with the drug's favorable safety profile, these results support the clinical benefits of long-term iptacopan treatment for patients with IgAN.

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. LB-24.

Sparsentan Beats Irbesartan for Proteinuria Reduction in FSGS Regardless of Baseline

Elevated proteinuria is associated with greater risk for progression to kidney failure among patients with focal segmental glomerulosclerosis (FSGS), whereas lower proteinuria is associated with lower risk across a range of UPCR thresholds.

Irbesartan and the dual endothelin-angiotensin receptor antagonist sparsentan can both reduce proteinuria in patients with FSGS. In the phase 3 DUPLEX trial, researchers compared sparsentan 800 mg/day with irbesartan at the maximum labeled dose of 300 mg/day. The 108-week randomized study included patients with FSGS without known secondary causes. Results showed that sparsentan led to a more rapid and sustained reduction in proteinuria compared with irbesartan.

In a post hoc analysis of DUPLEX data, **Jai Radhakrishnan, MD**, and colleagues compared the impact of sparsentan and irbesartan on clinically meaningful low proteinuria targets in patients with baseline UPCR of less than 3 g/g versus 3 g/g or greater. Their analysis assessed changes in UPCR and rates of UPCR of less than 0.3 g/g (complete remission of proteinuria), less than 0.7 g/g, and less than 1.5 g/g.

Sparsentan was linked with rapid and sustained proteinuria reductions, regardless of baseline UPCR. Patients receiving sparsentan in both baseline UPCR groups achieved complete remission and UPCR of less than 0.7 g/g and less than 1.5 g/g more often than those receiving irbesartan. Sparsentan was also well tolerated regardless of patients' baseline proteinuria.

These findings support the benefits of sparsentan over irbesartan among patients with FSGS across baseline proteinuria levels. This indicates that sparsentan could be the optimal treatment for achieving rapid and sustained proteinuria reductions, along with clinically meaningful low proteinuria thresholds, in patients with FSGS regardless of their proteinuria levels before initiation of treatment.

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. LB-23.



Can Atacicept Safely and Effectively Treat IgA Nephropathy?

The **cytokines** B-cell activating factor (BAFF) and A Proliferation-Inducing Ligand (APRIL) are central to the pathophysiology of IgAN. Therefore, atacicept, a native human TACI-Fc fusion protein that binds and inhibits these cytokines, has potential for treating IgAN.

In the ongoing double-blind, placebo-controlled, multinational phase 3 ORIGIN 3 trial, **Richard Lafayette, MD**, and colleagues are assessing atacicept's safety and efficacy in patients with biopsy-proven IgAN.

Participants were randomly assigned to receive either atacicept 150 mg, self-administered subcutaneously once a week at home, or placebo. The primary end point was the percentage change in UPCR from baseline from a 24-hour collection at 36 weeks. The researchers also evaluated galactose-deficient IgA1 (Gd-IgA1) percentage change from baseline, hematuria resolution, and safety.

The interim analysis included 203 participants, with 106 in the atacicept group and 97 in the placebo group. At baseline, 53.2% received stable sodium-glucose cotransporter 2 inhibitors (SGLT2i), with use balanced between groups.

Week 46 results show that atacicept was associated with a 45.7% UPCR reduction from baseline, compared with a 6.8% reduction for patients receiving placebo. This represents a statistically significant treatment difference of 41.8% (95% CI, 28.9%-52.3%; P<0.0001). In addition, SGLT2i use did not appear to influence atacicept's effect on proteinuria reduction.

Atacicept was also associated with significant improvements in Gd-IgA1 and hematuria. Adverse events occurred at a similar rate between groups, and most were mild or moderate.

Overall, these findings indicate that atacicept leads to significant reduction in proteinuria at 36 weeks compared with placebo. Combined with its favorable safety profile and additional Gd-IgA1 and hematuria benefits, these results support atacicept's use as a targeted disease-modifying therapy for patients with IgAN.

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. G-478.

Conference Coverage

New Orleans, Louisiana | May 6-10, 2026

Sibeprenlimab-Associated Infection Rates Comparable With Placebo

A prespecified interim analysis of VISIONARY, a phase 3 trial of sibeprenlimab in patients with IgAN, revealed an association between sibeprenlimab and a 51.2% ($P<0.001$) placebo-adjusted reduction in 24-hour UPCR at 9 months. At weeks 24 and 48, sibeprenlimab was associated with a decrease in serum immunoglobulin G (IgG) of 32% and 35%, respectively. The risk for infection remained comparable with that observed with placebo.

Jonathan Barratt, PhD, and colleagues presented results of analyses of hypogammaglobulinemia and infection risk with sibeprenlimab. The analyses used categories in the MedDRA v27.0 System Organ Class and CIOMS MedDRA Labeling Group to compare the incidence of infection with sibeprenlimab versus placebo. Post-baseline levels of IgG, collected every 4 weeks in the first 3 months and then every 12 weeks, were stratified by degree of hypogammaglobulinemia.

The percentages of patients experiencing infections/infestations were 49.0% (127 of 259) in the sibeprenlimab group and 45.0% (113 of 251) in the placebo group; rates of the most common infections were also comparable between the two groups.

There was no incidence of significant hypogammaglobulinemia (<300 mg/dL); 3.5% of patients (9 of 258) had an IgG level of less than 400 mg/dL. In the lower IgG strata, there was no increase in infection risk. Two patients with an IgG level of 600 mg/dL or greater had serious infections (including one patient who experienced one severe infection). One patient with an IgG level of 400-499 mg/dL experienced serious infection. There were no serious infections among patients with IgG less than 400 mg/dL.

In conclusion, the researchers wrote, "infection rates were comparable between sibeprenlimab and PBO [placebo]. In the sibeprenlimab arm, significant hypogammaglobulinemia was rare, reductions in IgG levels did not appear to increase the overall infection risk, and serious infections were uncommon across all IgG strata. Evaluation of 24-month safety data is planned."

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. G-444.

Reductions in Serum Uric Acid Sustained Over 48 Weeks in Stage 3 CKD

Among patients with uncontrolled gout (UG) and CKD, the efficacy of urate-lowering therapies may be limited. Nanoencapsulated sirolimus plus pegadricase (NASP) is an investigational, every 4-week sequential, two-component infusion therapy that reduces serum uric acid (sUA). The therapy consists of targeted nanoencapsulated sirolimus (NAS) and pegadricase.

Results of the phase 3 DISSOLVE I and II studies among patients with stage 3 CKD (CKD3) showed an association between NASP and a reduction in sUA levels plus additional improved outcomes through 24 weeks. **Nissreen Elfadawy, MD**, and colleagues reported results of a post hoc analysis of renal, safety, and efficacy outcomes through 48 weeks in patients in DISSOLVE I.

Patients were randomly assigned 1:1:1 to receive either a high-dose (HD: 0.15 mg/kg NAS) or low-dose (LD: 0.1 mg/kg NAS) NASP or placebo. A 4-week observation period followed each administration; the main study included six treatment periods. Patients were then eligible to enter a 24-week double-blind extension. The current analysis pooled data from the HD and LD arms (NASP group).

Of the 21 patients with CKD3 in DISSOLVE I, seven in the NASP group and six in the placebo group received all 12 doses. In both groups, median baseline blood urea nitrogen level, serum creatine level, and GFR remained generally stable over 48 weeks. Mean baseline sUA was 8.14 mg/dL in the NASP group and 8.05 mg/dL in the placebo group.

After the initial dose of NASP, mean sUA declined to 0.2 mg/dL and remained near baseline in the placebo group. Over 48 weeks, patients in the NASP group experienced a 96.3% reduction in cumulative sUA area under the curve versus placebo. Safety findings remained consistent with those previously reported for patients with CKD3.

In conclusion, the researchers wrote, "In patients with UG and CKD3, NASP stabilizes renal function and improves gout clinical outcomes over 48 weeks versus PBO [placebo]."

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. G-351.

Efficacy and Safety of Sibeprenlimab Unchanged by SGLT2i

In 2025, Kidney Disease: Improving Global Outcomes guidelines recommended add-on SGLT2i to address generic drivers of nephron loss in patients with IgAN. An interim analysis of data from VISIONARY, a phase 3 trial of sibeprenlimab among adults with IgAN, demonstrated the 9-month efficacy of sibeprenlimab in reducing 24-hour UPCR (UPCR-24h) across prespecified subgroups, including a subgroup with SGLT2i use at baseline.

Dana Rizk, MD, and colleagues presented results of an analysis designed to examine the impact of baseline use of SGLT2i on 24-hour proteinuria reduction, change in spot proteinuria over time, and safety and pharmacodynamics of sibeprenlimab.

Researchers stratified randomly assigned participants by use of SGLT2i at baseline and used analysis of covariance to evaluate UPCR-24h at 9 months. They used observed data to assess proteinuria and a mixed model for repeated measures to assess spot UPCR. They calculated observed mean percentage change from baseline for various antibodies as well as APRIL.

At baseline, characteristics were similar across subgroups. At 9 months, there was no change in the magnitude of UPCR-24h reduction with baseline SGLT2i use with sibeprenlimab or placebo. Baseline use of SGLT2i did not affect 12-month spot UPCR trajectory for either trial arm. The rates of achieving proteinuria response thresholds were comparable for both SGLT2i use and nonuse. SGLT2i status did not affect safety outcomes or pharmacodynamic markers.

"Sibeprenlimab led to similar clinically and statistically significant proteinuria reduction regardless of SGLT2i use at BL [baseline]," the researchers wrote. "These data support the use of sibeprenlimab independent of SGLT2i therapy."

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. G-488.



Patients Receiving Dialysis Benefit From Adjuvanted HBV Vaccine Formulation

Reduced or delayed response to vaccines presents a challenge in management of patients receiving long-term dialysis. Modified vaccination protocols in that patient population for hepatitis B virus (HBV) include use of higher-antigen doses, additional booster injections, or use of adjuvanted formulations.

Rachel Lasky, MPH, and colleagues reported results of an analysis examining HBV antigen response (antiHB) among a population of patients receiving dialysis who were administered adjuvanted HBV vaccine (aHBV-V) compared with those who received standard HBV vaccine (sHBV-V). Both groups received a four-dose initial regimen.

Eligible participants were patients at Fresenius Kidney Care who (1) were given an initial dose of aHBV-V between May 1, 2021, and April 30, 2025, or an initial dose of sHBV-V between May 1, 2015, and April 30, 2019; (2) had an antiHB concentration less than 10 IU/L before vaccination; (3) received four doses according to standard timing (aHBV-V within 30 to 120 days or sHBV-V within 270 days); and (4) had an antiHB concentration measured between 30 and 120 days after receipt of the final dose.

Vaccine response was determined by the antiHB closest to 60 days after the fourth dose. Patients with antiHB concentration less than 10 IU/L after the fourth dose were assessed again using the antiHB concentration obtained nearest to 6 months after the fourth dose, and the total doses received were noted.

After administration of four doses, 80.8% of those in the aHBV-V group were responsive (antiHB ≥ 10 IU/L), compared with 65.4% of those in the sHBV-V group. Nonresponsive patients (antiHB < 10 IU/L) were reassessed over the following 6 months. Nonresponsive patients in the aHBV-V group were more likely to have a response compared with those in the sHBV-V group (31.3% vs 25%, respectively). During the 6-month period, doses of aHBV-V and sHBV-V were given, on average, an additional 2.5 and 2.8 times, respectively.

"Among a large group of dialysis patients, patients vaccinated with aHBV-V were more likely to demonstrate an antiHB ≥ 10 IU/L than patients vaccinated with sHBV-V," the researchers wrote.

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. G-328.

Reducing Tolvaptan Fails to Cut Rapid Hyponatremia Correction Risk in SIADH

Tolvaptan is a vasopressin 2 receptor antagonist that can treat hyponatremia in the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Some patients receiving tolvaptan experience a rapid correction of hyponatremia, leading to the risk of overcorrection and making many physicians reluctant to prescribe the drug.

Lowering the dose of tolvaptan is one possible approach for minimizing risk. **Pooja Punukollu** and colleagues assessed the effectiveness of approach among 160 hospitalized adult patients diagnosed with SIADH and treated with tolvaptan between 2017 and 2025. The researchers excluded patients with eGFR of less than 30 mL/min/1.73 m², heart failure, or cirrhosis and those who did not have a follow-up serum sodium measurement within 24 $\pm$ 2 hours of the baseline value measurement before tolvaptan initiation. Median serum sodium, serum osmolality, urine sodium, and urine osmolality values were 124 mEq/L, 270 mOsm/kg, 75 mEq/L, and 468 mOsm/kg, respectively.

One hundred twenty-six patients (79%) received a 15-mg dose, and the other 34 (21%) received a reduced dose of 7.5 mg. Rates of adequate correction, defined as a 4 mEq/L or greater rise in 24 hours, were similar between the two groups [61.7% for the 7.5-mg dose group and 68.0% for the 15-mg dose group; $P=0.49$]. Similar results occurred on comparison of rates of rapid correction, defined as 10 mEq/L or greater in 24 hours (14.7% for the 7.5-mg dose group and 9.5% for the 15-mg dose group; $P=0.38$).

These results indicate that lowering the starting dose of tolvaptan does not reduce a patient's risk for rapid hyponatremia correction. However, the authors note that efficacy also did not decrease, suggesting that patients can be effectively treated at a reduced dosage. Further studies could compare tolvaptan 7.5 mg with 3.75 mg to determine whether rapid correction is reduced when the dose is lowered further.

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. G-408.



Pegcetacoplan Sustains Proteinuria Reduction Across Diverse IC-MPGN Populations

In the 26-week randomized controlled period of the phase 3 VALIANT trial, the C3/C3b inhibitor pegcetacoplan led to significant reductions in proteinuria among patients with immune complex membranoproliferative glomerulonephritis (IC-MPGN) when compared with placebo.

In the following 26-week open-label period, all patients received pegcetacoplan, including 59 of 63 from the original pegcetacoplan group and 55 of 61 from the original placebo group. Efficacy end points included changes from baseline in proteinuria and eGFR, with TEAEs also noted.

Findings presented by **Gashu Ayehu, MD**, showed a sustained reduction in proteinuria among patients treated with pegcetacoplan over 52 weeks. At week 26, proteinuria decreased by a mean of 67.2% (95% CI, -74.9% to -57.2%), and this reduction was maintained at week 52 (-67.2%; 95% CI, -75.8% to -55.4%). Results were consistent across subgroups, including patients with and without immunosuppressant use, those with baseline nephrotic-range proteinuria (≥ 3 vs < 3 g/g), and adolescents aged 12 to 17 years. Kidney function remained stable overall in the pegcetacoplan-treated population, with least squares mean eGFR changes of -1.5 mL/min/1.73 m² (standard error [SE], 2.2) at week 26 and -3.7 mL/min/1.73 m² (SE, 2.7) at week 52. No new safety signals were identified.

Overall, these results confirm that pegcetacoplan has sustained and consistent efficacy in patients with IC-MPGN regardless of immunosuppressant use, baseline proteinuria, or patient age.

REFERENCE

2026 National Kidney Foundation Spring Clinical Meetings. Abstract No. G-427.

Conference Coverage

New Orleans, Louisiana | May 6–10, 2026

Awards and Honors

Healthcare professionals who have made significant contributions to the field of kidney disease were honored at the National Kidney Foundation (NKF) 2026 Spring Clinical Meetings.



Katalin Susztak, MD, PhD

The David M. Hume Memorial Award, the highest honor given by the NKF to a scientist-clinician in the field of kidney and urologic diseases, went to **Katalin Susztak, MD, PhD**, professor of medicine and genetics at the University of Pennsylvania and founding director of the Penn-CHOP Kidney Innovation Center. Her research explores the genetic and molecular mechanisms associated with kidney disease to inform development of precision therapeutics.



Mona Calvo, PhD

Mona Calvo, PhD, received the Joel D. Kopple Distinguished Lecture Award for significant contributions to the field of renal nutrition. Dr. Calvo spent many years working at the FDA's Center for Food Safety and Applied Nutrition and is currently an adjunct research professor in the Renal Division at the Icahn School of Medicine at Mount Sinai in New York City. Her research focuses on the effects of diet on hormonal regulation of calcium, phosphorus, and vitamin D and their impact on the prevention and management of bone and kidney disease.



Ron Wald, MDCM, MPH

Ron Wald, MDCM, MPH, medical director of hemodialysis at St. Michael's Hospital in Toronto, Canada, and faculty at Tel Aviv Sourasky Medical Center, Israel, received the J. Michael Lazarus Distinguished Award, honoring nephrologists whose work advances dialysis care and renal replacement therapy research. Dr. Wald's research spans critical care nephrology and maintenance dialysis, including leadership in international trials such as STARRT-AKI, Probe-Fluid, WISDOM, and PHOSPHATE, shaping patient care worldwide.



Roslyn Mannon, MD

This year's Excellence in Kidney Transplantation Award recipient was **Roslyn Mannon, MD**. She is a professor at the University of Nebraska Medical Center, where she also serves as vice chair for research for the Department of Internal Medicine and associate chief of research in the Division of Nephrology. Dr. Mannon has published widely on chronic allograft failure and posttransplant complications and therapeutics.



Brad Rovin, MD

Brad Rovin, MD, received the Donald W. Seldin Award in recognition of excellence in clinical nephrology. Dr. Rovin, a professor of medicine and pathology at the Ohio State University Wexner Medical Center and the Lee A. Hebert Professor of Nephrology, focuses on the immunopathogenesis of glomerular and autoimmune diseases and biomarker development for noninvasive assessment of kidney pathology.



Carol Kirila, DO

Carol Kirila, DO, was the recipient of the Medical Advisory Board Distinguished Service Award, highlighting community service and activities that promote the NKF's mission on a local level. Dr. Kirila is an internal medicine physician and an adjunct professor of internal medicine in the Department of Primary Care in the College of Osteopathic Medicine at Kansas City University. She chairs the Medical Advisory Board for the National Kidney Foundation of Western Missouri, Kansas, and Oklahoma, and is a member of the NKF Health Equity Committee.



Sylvia E. Rosas, MD, MSCE

The Garabed Eknoyan Award, given in recognition of an individual whose work promotes the mission of the NKF in making lives better for people with kidney disease, went to **Sylvia E. Rosas, MD, MSCE**. Dr. Rosas is a nephrologist and physician-scientist at the Joslin Diabetes Center and the Beth Israel Deaconess Medical Center in Boston, Massachusetts, and the director of the Latino Kidney Clinic and an associate professor of medicine at Harvard Medical School. Her research focuses on the epidemiology of chronic kidney disease with an emphasis on diabetic kidney disease.



Tina Alexander, RN

The Carol Mattix Award, named for a home dialysis training nurse whose tireless work improved the lives of kidney patients, was given to **Tina Alexander, RN**. Alexander's 42 years of nursing experience, including 11 years in dialysis and 5 years in homes, have informed her work, teaching patients the skills they need to live a full life, helping them understand treatment options, and empowering them to be in control of their health.



Ann O'Hare, MD

The Shaul G. Massry Distinguished Lecture Award went to **Ann O'Hare, MD**, a nephrologist at the VA Puget Sound Health Care System in Seattle and professor of medicine at the University of Washington, where she is the inaugural holder of the Bill Peckham Endowed Fund for Person-Centered Care. She has spent her career developing approaches to providing compassionate care for people with kidney disease.



Lindsey Fuller, MS

The Celeste Castillo Lee Patient Engagement Award was presented to **Lindsey Fuller, MS**, who was diagnosed with familial C3 glomerulopathy (C3G) in 2013 at the age of 33. Ms. Fuller created C3G Warriors, an online community providing connection, information, and hope for those affected by C3G. She serves on the NKF Kidney Advocacy Committee and collaborates with other advocacy organizations and pharmaceutical companies to ensure inclusion of patient perspectives in shaping research and care. ●



Staying the Course

What clinicians can do to support immunosuppression adherence after kidney transplantation

By Charlotte Robinson

Long-term success in kidney transplantation depends on more than surgical skill or advances in immunosuppressive therapy. Medication adherence—shaped by clinical, behavioral, and system-level factors—remains one of the most enduring challenges to graft survival.

Maria C. Litchfield, MSN, APRN, AGACNP-BC, senior medical science liaison—abdominal at Thermo Fisher

Scientific, has spent years caring for transplant recipients as a coordinator, nurse practitioner, and clinical operations leader. Across those roles, she has seen similar barriers emerge time and again.

“Number 1 is the amount of pills that these transplant patients have to take, especially at the beginning when they are first posttransplant,” she told *Nephrology Times*. The side effects of immunosuppressive drugs, which pose a

challenge to adherence, are often managed with additional medications, adding to the daily pill burden.

Cost is another persistent hurdle. Despite pretransplant counseling, patients may feel overwhelmed by out-of-pocket expenses. “We say yes to everything at the beginning, but then afterward we really feel that burden of cost that comes with immunosuppression medications,” Litchfield said. Gaps in sustained education can compound all these barriers, particularly when patients fail to appreciate how missed doses or delayed laboratory tests affect long-term graft survival.

“Overall, transplant centers do an excellent job of educating their patients and getting the point across. I think where the gap may be is that we don’t do it enough,” Litchfield said. “It’s really difficult to absorb an enormous amount of information in one sitting and keep it for the long term.” She advocates for smaller, repeated educational touchpoints that reinforce the role of immunosuppression, the risks for infection and rejection, and the importance of consistent monitoring. Every interaction—whether electronic, virtual, or in person—offers an opportunity for reinforcement.

For busy clinicians, identifying adherence risks requires leaning on the broader transplant team. Litchfield emphasized the importance of coordinators and medical assistants, who maintain frequent contact with patients and often detect



problems early. Regular team check-ins create space to discuss barriers, share observations, and develop practical solutions rooted in real-world patient experience.

When patients miss labs or appointments, it is usually due to competing responsibilities or limited understanding of consequences, Litchfield said. Reducing logistical burdens through local laboratory testing and satellite clinics can help. Reengagement begins with listening, followed by mobilizing multidisciplinary resources—including social workers and financial coordinators—to address cost, transportation, or caregiving challenges.

“I think that most patients are very grateful to have received a transplant and overall want—to the best of their ability—to take care of the graft,” Litchfield said. ●



Maria C. Litchfield, MSN, APRN, AGACNP-BC

Scan the code
to watch our
interview with
Maria Litchfield.



Excluded by Design

Why kidney transplant recipients deserve a place in clinical trials

By Keightley Amen, BA, ELS



Clinical trials are designed to determine whether a drug or treatment is effective under ideal, highly controlled conditions. However, these strict protocols often require excluding the very patients who will ultimately use the therapies in real-world clinical settings. As a result, our understanding of broader safety and effectiveness in typical patient populations is limited.

This challenge is evident across many disease areas, including transplantation, and kidney transplant recipients are frequently excluded from trials evaluating treatments for conditions such as cardiovascular disease, diabetes, and infections.

Many experts are advocating for increased inclusion of transplant recipients, a growing population that requires lifelong medical therapy for a variety of health issues. In addition, advocates argue that inclusion will help clinicians make better-informed treatment decisions, rather than extrapolate evidence from studies that involve only nontransplant populations. And, importantly, their inclusion will help ensure payers cover treatments for transplant recipients.

“Transplant recipients have a high burden of comorbid disease and may benefit from new therapies. Their inclusion allows for a better understanding of safety and effectiveness in a population that is frequently treated but understudied,” **Krista L. Lentine, MD, PhD**, a professor of medicine at Saint Louis University in Missouri and the Mid-America Transplant/Jane A. Beckman Endowed Chair in Transplantation, told *Nephrology Times*. “In addition, excluding transplant recipients affects the generalizability of trial results. Trials that exclude patients on immunosuppression or with complex medical conditions may not reflect real-world populations.”

The Practical Reasons for Exclusion

Exclusion of transplant recipients is a result of a combination of structural, scientific, and regulatory reasons, rather than a single barrier, Dr. Lentine explained. Immune status is a common barrier.

“Sometimes, trial protocols are written with exclusion criteria that preclude transplant patients through language like ‘immunocompromised’ or ‘on chronic immunosuppression.’ That’s not always intentional, but it effectively removes transplant patients from participation,” she explained.

“There’s also a degree of risk aversion. Sponsors and investigators may be concerned about safety signals in a more complex population, potential drug-drug interactions with immunosuppressive regimens, or the risk of allograft dysfunction. From a trial design perspective, transplant recipients are often seen as introducing heterogeneity that could complicate the interpretation of results.”

There is a reasonable basis for such exclusion criteria, according to **Peter Heeger, MD**, professor of medicine, surgery, and biomedical sciences at Cedars-Sinai Medical Center in Los Angeles, California. As Dr. Heeger explained to *Nephrology Times*, “Many people would say that transplant patients are different, so the risks are different and the mechanisms are different. So, if you’re testing a new drug for hypertension, it may not work on the mechanism. These are very rational reasons, as the pharmacology is significant.”

Other challenges to broader inclusion include detailed practical research considerations. For example, transplant recipients require close monitoring, may require different study end points, and require care at specialized centers, all of which make trial implementation even more complex.

In addition, Dr. Heeger noted that transplant recipients may be wary of enrolling in clinical trials. “One of our current clinical trial barriers is hesitancy to enroll in clinical trials, questions about it being too risky, or moving away from the standard of care. People have said they don’t want to be ‘guinea pigs,’” he said. Transplant recipients may be even more hesitant because of the medical issues they have already endured.

Another logistical challenge for transplant recipients is that they may not be up to date on vaccinations, which is a common criterion in clinical research, Dr. Heeger added.

Exclusion criteria may even affect inclusion in studies specifically involving renal transplant recipients. In a 2018 study titled “Do Clinical Trials Reflect Reality? A Systematic Review of Inclusion/Exclusion Criteria in Trials of Renal Transplant Immunosuppression,” **Anam A. Ayaz-Shah, PhD**, and colleagues extracted data regarding inclusion and exclusion criteria from 174 trials of immunosuppression in renal transplant recipients and compared the criteria with those specified in published trial registries. Frequently reported donor exclusion criteria included age, donor type, and cold ischemic time. Common recipient exclusion criteria included upper age limit, high panel reactive antibody, and previous transplantation (39.7%).

The implications of excluding transplant recipients can be significant for patients and families.

The authors wrote, “Our findings suggest many recent immunosuppression trials have restrictive inclusion criteria which may not be reflective of current renal transplant populations. Discrepancies between trial protocols and published reports raise the possibility of selection bias.”

The Implications of Exclusion for Patients and Families

The implications of excluding transplant recipients can be significant for patients and families. When this population is excluded, there is insufficient evidence to guide treatment decisions. This may delay adoption of new therapies; clinicians may be hesitant to use a therapy because safety and efficacy have not been established in patients receiving immunosuppression.

There are also downstream effects on regulatory approval and insurance coverage. When transplant recipients are not included in clinical trials, indications may not extend to them; therefore, payers may deny coverage or require additional justification. That creates barriers to access even when a therapy may be clinically appropriate.

“For patients and families, this translates into uncertainty and variability in care. Decisions may differ across centers or providers, and patients may not have access to the same treatments as others with similar conditions,” Dr. Lentine said.

“Ultimately, this can affect outcomes. Delayed or inconsistent access to effective therapies may impact disease control, complications, and quality of life.”

The Implications of Exclusion for Healthcare Professionals

For healthcare professionals, the issue creates uncertainty in clinical decision-making.

“In the absence of trial data specific to transplant recipients, clinicians often need to extrapolate from nontransplant populations when managing common conditions such as cardiovascular disease, diabetes, and infections. That approach is necessary, but it is not ideal, particularly in patients with altered physiology and chronic immunosuppression,” Dr. Lentine said. “Questions related to safety, dosing, drug-drug interactions, and impact on allograft function are often not directly addressed in the available evidence. As a result, treatment strategies may vary across providers and centers.”

She called specific attention to cardiometabolic disease and cardiovascular care, which significantly affect transplant recipients. Despite the higher risk in transplant recipients, there are gaps in evidence, and they limit healthcare professionals’ ability to apply even well-established therapies with confidence in this particular population.

The issue also has implications for guideline development and standardization of care, as well as difficulty obtaining coverage for off-label therapies.

The Steps to Improve Inclusion

Dr. Lentine said that the field recognizes the issue, but it is still early in terms of translating that recognition into widespread changes in trial design or eligibility criteria.

Although the reasons for exclusion may be understandable, given the complexity of clinical research, Dr. Lentine and other advocates argue that they are structural and worth overcoming.

“The consequence is a population that is systematically underrepresented, despite having a high disease burden and frequent exposure to the therapies being studied,” she said. “To generate evidence that applies to transplant recipients, sponsors need to be more intentional about including them, rather than excluding them by default.”

She recommended several ways to adjust and evolve trial design to better include transplant recipients.

- Move away from blanket exclusions based on transplant status or immunosuppression. Instead, use more specific, risk-based criteria. “Not all transplant recipients carry the same level of risk, and eligibility can reflect that,” Dr. Lentine said.
- Incorporate dedicated transplant cohorts or stratified enrollment, allowing inclusion without compromising interpretability of results. This would create an opportunity to generate transplant-specific safety and efficacy data within the same study.
- Build protocols that give intentional attention to drug-drug interactions and dosing strategies, particularly with immunosuppressive regimens, rather than use them as reasons for exclusion.

- Develop enhanced monitoring frameworks for participants with transplants, such as closer follow-up or predefined safety end points related to allograft function.
- Collaborate with transplant centers and specialists during trial design to ensure that protocols are both practical and clinically relevant for this population.
- Expand the population studied. “We might include more people in the trial by relaxing the criteria, but then the trial needs to be larger in order to have better statistical power,” Dr. Heeger said. This can be challenging, however, because “transplantation is an orphan disease, so we may not have the number of people to apply some of the strategies and approaches as oncology trials.”

“These changes do not eliminate complexity, but they make inclusion more achievable and allow for evidence that is more applicable to this important and vulnerable patient population,” Dr. Lentine concluded.

How to Advocate for Transplant Recipients

Nephrologists and transplant clinicians play a key role in advocating for inclusion and identifying appropriate trial candidates.

“At a minimum, we should be raising awareness of the gap, both in clinical practice and in academic settings, so that it is recognized as a limitation in how we generate and apply evidence,” Dr. Lentine said.

Nephrologists and transplant clinicians can also lend their expertise by contributing to trial design as investigators, collaborators, or reviewers. This would put them in a position to advocate for more thoughtful inclusion criteria, rather than default exclusions, or to encourage special cohorts or subgroup analyses.

Professional societies can also play a major role in the inclusion of more transplant recipients in clinical trials. For example, they can develop advisory groups and guidelines to encourage and support more inclusive research practices.

Finally, nephrologists and transplant clinicians can conduct their own research, documenting and publishing their own observed outcomes in transplant populations, which can inform and influence future research.

Normalizing inclusion of transplant recipients only stands to strengthen evidence-based care, ensuring that new therapies are evaluated in the patients who will ultimately receive them.

“We want our transplant patients to live normal, healthy, long lives; and there are so many things about posttransplant care that are complicated, such as graft loss, immune system, polypharmacy, comorbidities, high blood pressure, and more,” Dr. Heeger said. “Anything we can do to include these patients and improve their health is preferential.” ●

REFERENCE

Ayaz-Shah AA, et al. *Transpl Int*. 2018;31(4):353-360. doi:10.1111/tri.13109

What's your go-to approach for managing resistant hypertension in CKD?



"Shared decision-making and close monitoring are key. Balancing BP reduction, cardiovascular risk, and slowing CKD progression, I individualize therapy based on estimated glomerular filtration rate (eGFR), potassium, albuminuria, and progression risk. My approach prioritizes long-acting angiotensin II receptor blockers with thiazide-like diuretics. I add potassium binders earlier for hyperkalemia and use loop diuretics for eGFR below 25 mL/min/1.73 m² or persistent volume overload. Calcium channel blockers are typical third-line agents; mineralocorticoid receptor antagonists (MRAs) are introduced earlier for persistent proteinuria if potassium permits. I reach for nonsteroidal MRAs and glucagon-like peptide-1 agonists sooner for diabetic patients. I also screen all patients with hypertension for primary aldosteronism."

Jehan Zahid Bahrainwala, MD
Stanford University School of Medicine



"Resistant hypertension in CKD is common and rarely solved by adding another agent blindly. My approach starts with confirming the diagnosis using out-of-office measurements and reviewing adherence and dietary sodium, which is often the biggest lever. I optimize a three-drug foundation—an ACE [angiotensin-converting enzyme] inhibitor or ARB [angiotensin receptor blocker], a long-acting calcium channel blocker (nifedipine), and a thiazide-like diuretic (chlorthalidone)—before escalating. For true resistance, I add spironolactone when potassium and eGFR allow, with close monitoring. I also screen for secondary causes, particularly primary aldosteronism and OSA [obstructive sleep apnea], which are underdiagnosed in this population."

Katalin Susztak, MD, PhD
Perelman School of Medicine at the University of Pennsylvania



"Go through all the patient's medications and doses in detail to assess for adherence or any obstacles. Make sure they actually fit the criteria of resistant hypertension (often they are not on multiple medications or maximum doses). Check their technique of BP measurement, review home log and monitoring (24-hour ambulatory BP monitoring, if available, is even better), and review diet and lifestyle factors. Start a workup of secondary causes based on their history and exam, including but not limited to comprehensive metabolic panel, laboratory analysis for renin and aldosterone levels, results of ultrasound and other necessary imaging, and metanephrine, thyroid, and parathyroid analysis, and cater treatment to the findings."

Samaya J. Anumudu, MD
Baylor College of Medicine



"Treatment of resistant hypertension in CKD starts with evaluation of adherence to therapy, screening for secondary causes (particularly primary aldosteronism) and interfering substances, and accurate measurement of blood pressure out of office (automated home monitoring or ABPM [ambulatory blood pressure monitoring]). Optimize volume with a thiazide-like diuretic (chlorthalidone 12.5 to 25 mg) plus an ACE or ARB plus a CCB [calcium channel blocker]. If eGFR is greater than 40 mL/min/1.73 m² and potassium allows, add spironolactone 12.5 to 25 mg daily with close monitoring. If spironolactone is contraindicated, a combination of minoxidil and clonidine (optimize diuretics!) or carvedilol is next. Evaluate volume and retest adherence." ●

Susan Steigerwalt, MD
University of Michigan (retired)



New Leadership Takes the Helm at Two Nephrology Journals

Two leading nephrology journals are preparing for new leadership. The International Society of Nephrology has named **Jai Radhakrishnan, MD**, as the next editor-in-chief of *Kidney International*, effective January 1, 2027. A Columbia University professor and current editor of *Kidney International Reports*, he succeeds **Pierre Ronco, MD, PhD**, and aims to build on the journal's legacy while advancing high-impact global research.

Similarly, the National Kidney Foundation has appointed **Haewook Han, PhD, RD, LDN**, as editor-in-chief of the *Journal of Renal Nutrition*, also starting January 1, 2027. A Tufts University leader and renal nutrition expert, Dr. Han will transition into the role alongside outgoing editor **Linda Moore, PhD, RD**.

SOURCES: INTERNATIONAL SOCIETY OF NEPHROLOGY,
NATIONAL KIDNEY FOUNDATION

AKF Launches Professional Education Curriculum to Address Kidney Care Gaps

The American Kidney Fund (AKF) has launched a 2-year professional education curriculum aimed at interdisciplinary healthcare professionals involved in kidney disease prevention, detection, and care coordination. The initiative is intended to address knowledge gaps among frontline clinicians and support earlier detection and improved management.

The program will deliver eight quarterly continuing education and CME-accredited activities over 2 years. It is designed for nurses, nurse practitioners, physician assistants, social workers, dialysis technicians, and certified health education specialists. Content will progress from foundational topics in the first year to more advanced, patient-centered subjects in the second.

SOURCE: AMERICAN KIDNEY FUND



FDA UPDATES



FDA Approves First Generic Dapagliflozin Tablets

The FDA approved the first generic versions of dapagliflozin tablets, expanding access to a sodium-glucose cotransporter 2 inhibitor widely used for patients with type 2 diabetes (T2D), including those with cardiovascular risk factors.

The generic versions of dapagliflozin are approved to reduce the risk of hospitalization for heart failure in adults with T2D who have established cardiovascular disease or multiple cardiovascular risk factors. They are also approved as an adjunct to diet and exercise to improve glycemic control in adults with T2D.

The approvals reference the same labeled indications as the branded drug, and the prescribing information for the generic formulations carries the same contraindications, warnings, and precautions.

SOURCE: US FOOD AND DRUG ADMINISTRATION

Baxdrostat Approved by FDA for Hypertension

The FDA approved baxdrostat for use in combination with other antihypertensive agents in adults with inadequately controlled hypertension. Approval was based on results of the phase 3 BaxHTN trial, which enrolled 796 patients with uncontrolled or resistant hypertension receiving at least two background therapies, including a diuretic. At 12 weeks, baxdrostat produced statistically significant reductions in seated systolic BP versus placebo at 2-mg and 1-mg doses.

Baxdrostat selectively inhibits aldosterone synthase (CYP11B2), lowering aldosterone levels without affecting cortisol in clinical studies. Further investigation is ongoing in conditions linked to elevated aldosterone, including CKD and primary aldosteronism.

SOURCE: ASTRAZENECA

Povetacicept Advances to FDA Review in IgAN

The FDA accepted Vertex Pharmaceuticals' biologics license application for povetacicept for accelerated approval in adults with IgA nephropathy (IgAN). The Prescription Drug User Fee Act target action date is November 30, 2026.

The submission is supported by a prespecified 36-week interim analysis from the phase 3 RAINIER trial, in which povetacicept treatment was associated with a 52.0% reduction from baseline in urine protein-to-creatinine ratio, representing a 49.8% reduction compared with placebo ($P=0.001$). Reductions in proteinuria were consistent across prespecified subgroups.

Povetacicept is an investigational BAFF (B-cell activating factor) and APRIL (a proliferation-inducing ligand) inhibitor. ●

SOURCE: VERTEX



CHRONIC KIDNEY DISEASE

Should RAS Inhibition Be Discontinued After an Acute eGFR Decline?

JAMA Netw Open. 2026 Mar 2;9[3]:e263680. doi:10.1001/jamanetworkopen.2026.3680

Renin-angiotensin system (RAS) inhibitors slow CKD advancement but are often discontinued if acute decreases in estimated glomerular filtration rate (eGFR) occur after initiation. **Elaine Ku, MD**, and colleagues questioned whether this practice is optimal and compared the kidney, cardiovascular, and mortality risks linked to discontinuing versus continuing RAS inhibitors in this scenario.

Researchers conducted the retrospective cohort study, leveraging electronic records from the Manitoba Centre for Health Policy in Canada. They applied propensity score matching to pinpoint adults (aged ≥ 18 years) who were given new prescriptions for RAS inhibitors and experienced an eGFR decrease of more than 15% within 90 days. The participants received the medications between January 1, 2008, and December 31, 2021, and the investigators analyzed the data between January 14, 2024, and January 22, 2026. Discontinuation versus continuation was determined based on whether participants refilled their prescription after 90 days following a 15% decline in eGFR. Cox proportional hazard models were used to compare exposure with outcomes.

Primary outcomes were end-stage kidney disease (ESKD) or death. Secondary outcomes were acute kidney injury (AKI) or major adverse cardiovascular events (MACE) occurring within 180 days.

Among 4,233 participants, 1,411 (33.3%) discontinued RAS inhibitors, and 2,822 (66.6%) continued them. Those who discontinued RAS inhibitors had a higher risk for ESKD (hazard ratio [HR], 1.74; 95% CI, 1.28-2.38) and death (HR, 1.23; 95% CI, 1.07-1.41). However, the patients who discontinued RAS inhibitors did not have a significantly higher risk for AKI (HR, 1.11; 95% CI, 0.90-1.37) or MACE (HR, 1.13; 95% CI, 0.99-1.28).

Since continuing RAS inhibitors is linked to a lower risk for ESKD and death, future research should explore the reasons for frequent discontinuation and identify strategies to prolong the use of RAS inhibitors, the authors concluded.

Empagliflozin-Finerenone Albuminuria Reduction Is Consistent Across Age and Sex

Nephrol Dial Transplant. doi:10.1093/ndt/gfag075

Concurrent initiation of empagliflozin and finerenone can reduce albuminuria more than the individual therapies at 180 days among individuals with CKD and type 2 diabetes. In an analysis of CONFIDENCE trial data, **Rajiv Agarwal, MD**, and colleagues assessed how sex and age can influence the ability of the medications to lower urine albumin-to-creatinine ration (UACR).

CONFIDENCE included participants with type 2 diabetes, albuminuria (UACR 100 to $< 5,000$ mg/g), and CKD (eGFR 30-90 mL/min/1.73 m²) who were receiving stable regimens of RAS inhibitors. Investigators randomly assigned the participants 1:1:1 to receive finerenone 10 or 20 mg/day, empagliflozin 10 mg/day, or both. The current analysis stratified participants by sex and by age groups (≤ 60 years, 61-68 years, 69-74 years, > 74 years).

Outcomes included the percentage change in UACR from baseline to 180 days and the occurrence of key adverse events.

Increasing age was linked to a higher baseline occurrence of atherosclerotic cardiovascular disease and lower eGFR. Older age significantly predicted UACR reduction in all three groups, with a -10.2% (95% CI, -15.5 to -4.6) incremental change each 10-year age increase at day 180 (P for age effect=0.001). Women displayed an 18.6% (95% CI, 6.3-29.2) larger UACR reduction at 180 days than men (P for sex effect=0.008).

The occurrence of adverse events was similar between men and women and across age groups. Researchers noted no interaction between sex (P -interaction=0.46) or age (P -interaction=0.28) on the effect of combination therapy.

The findings indicate that the UACR-lowering response of empagliflozin and finerenone scaled linearly with sex (~20% more among women) and age (10% per decade). Combination therapy produced benefits that were independent of sex and age.

Kidney Protection and Survival With Semaglutide by CKD Severity in the FLOW Trial

Clin J Am Soc Nephrol. doi:10.2215/CJN.0000000974

The FLOW trial found that semaglutide reduced the risk of kidney disease events and improved overall survival among participants with type 2 diabetes and CKD. In a posthoc analysis, **Katherine R. Tuttle, MD**, and colleagues aimed to determine how these benefits differ across a broad range of CKD severity.

FLOW, which was a double-blind, randomized, placebo-controlled trial, had a median follow-up time of 3.4 years. Some participants had type 2 diabetes and an eGFR of 50-75 mL/min/1.73 m² and UACR of greater than 300 but less than 5,000 mg/g. Other participants had type 2 diabetes and an eGFR of 25 to less than 50 mL/min/1.73 m² and UACR of greater than 100 but less than 5,000 mg/g.

The primary outcome was a composite of factors including eGFR of less than 15 mL/min/1.73 m², 50% or greater eGFR decline, dialysis, kidney transplant, and fatalities due to cardiovascular or kidney causes, in addition to all-cause fatalities. Investigators randomly assigned participants to receive subcutaneous semaglutide 1 mg once per week or a placebo. They categorized participants into subgroups based on baseline eGFR (< 30 to ≥ 60 mL/min/1.73 m²) or UACR (< 100 to $\geq 2,000$ mg/g) to evaluate the primary outcome.

The primary outcome occurred in 19% of participants receiving semaglutide treatment and 23% of participants receiving the placebo (HR, 0.76; 95% CI, 0.66-0.88). Fatalities occurred in 13% and 16% of those in the semaglutide and placebo groups, respectively. Across UACR and eGFR subgroups, HRs for the primary outcome were consistent. For fatalities, although the HRs were very similar among eGFR subgroups, the HR was lowest for those with a UACR of 2,000 mg/g or greater.

The researchers concluded that the results support semaglutide treatment for patients with type 2 diabetes with all levels of CKD severity.



Finerenone Improves Kidney Function in Type 1 Diabetes

N Engl J Med. 2026;394(10):947-957. doi:10.1056/NEJMoa2512854

Although earlier research has shown that finerenone improves kidney and cardiovascular outcomes in patients with type 2 diabetes and CKD, use of the medication has not been evaluated in people with type 1 diabetes and CKD. To address the gap, **Hiddo J. L. Heerspink, PhD**, and colleagues examined the use of finerenone in this population and determined that it is effective.

Their phase 3 clinical trial included 242 adults with type 1 diabetes, CKD (eGFR of 25 to <90 mL/min/1.73 m²), and albuminuria (UACR of 200 to <5,000 mg/g). Participants received either an angiotensin receptor blocker or an angiotensin-converting enzyme inhibitor. Investigators randomly assigned them to receive finerenone (10 or 20 mg per day, depending on the eGFR) or a placebo. The primary outcome was the relative change in UACR over 6 months.

The UACR was reduced from 574.6 mg/g at baseline to 373.5 mg/g at 6 months among participants who received finerenone, and from 506.4 mg/g to 475.6 mg/g among those who received placebo. This indicates that finerenone produced a 25% larger reduction than the placebo.

In addition, at 6 months, the finerenone group experienced an eGFR change of -5.6 mL/min/1.73 m², while the placebo group experienced a change of -2.7 mL/min/1.73 m², a difference of 2.9 mL/min/1.73 m².

The most frequent adverse event was hyperkalemia, occurring in 10.1% of participants in the finerenone group and in 3.3% of participants receiving placebo, with 1.7% requiring discontinuation for this reason.

The researchers concluded that finerenone produced a significantly larger reduction in UACR than the placebo.

GLOMERULAR DISEASES

Comparison of UACR and UPCR in Predicting Kidney Failure

Nephrol Dial Transplant. doi:10.1093/ndt/gfag066

Proteinuria is linked to long-term risk for kidney failure, and clinical guidelines around diabetic kidney disease and CKD favor UACR for measuring proteinuria. However, research on rare glomerular diseases frequently uses urine protein-to-creatinine ratio (UPCR) to measure proteinuria. In a new study, **Katie Wong** and colleagues explored whether UACR, UPCR, or other proteinuria measurements differ in their ability to predict kidney risk.

The study included patients in rapid assessment for disease and risk with same-day UACR and UPCR measurements. Investigators performed analyses on the whole cohort, as well as by UACR tertile and disease subtype. They used calibration plots to evaluate the performance of converting UPCR to UACR, and they ran Cox regression models of time to kidney failure or death with four proteinuria measures: UPCR, UACR, non-albumin proteinuria, and estimated UACR from UPCR. The research team also assessed model fit with Schwarz Bayesian information criterion (SBC) values and Akaike information criterion (AIC), as well as receiver operating characteristic curves and time-dependent area under the curve (AUC).

Among the cohort of 4,156 patients with rare kidney diseases, 17% experienced kidney failure or death. At lower predicted UACR deciles, the change from UPCR to UACR resulted in underestimations of UACR. The researchers noted very comparable SBC and AIC for all proteinuria measurements. Time-dependent AUCs at 5 years were greater than 0.83 for all disease subgroups and models. No statistically significant differences in model concordance were observed. Within each UACR tertile, SBC, AIC, and AUC values were similar across all proteinuria metrics.

Analyses showed no statistically significant differences in the performance of the proteinuria measurements, suggesting that none is superior to the others for predicting kidney failure or death.

HYPERTENSION

Baxdrostat Reduces Seated BP in Resistant Hypertension

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Aldosterone regulation is a key factor in the pathogenesis of hard-to-control hypertension. Although prior studies have shown that selective aldosterone synthase inhibitors reduce BP measured in a clinician's office, they haven't tested the medications through 24-hour ambulatory monitoring. Since the latter type of BP measurement may provide a more accurate assessment, **Michel Azizi, MD**, and colleagues evaluated the effect of baxdrostat, a selective aldosterone synthase inhibitor, in this manner among participants with resistant hypertension.

Bax24 was an international, phase 3, randomized, double-blind, placebo-controlled trial that included adults from 79 clinical sites in 22 countries. The participants had seated BP (SBP) within a range from 140 mm Hg or higher to less than 170 mm Hg, even though they were taking at least three hypertensive drugs. After a 2-week placebo period, investigators assigned participants with 24-hour ambulatory SBP of 130 mm Hg or higher 1:1 to receive 2 mg baxdrostat or a placebo once daily for 12 weeks, along with background therapy. They also performed a safety analysis on those who received at least one dose of medication.

Of the 217 participants (baxdrostat: n=108; placebo: n=109), 65% were male, 78% were white, and the median age was 60 years. At 12 weeks, the change from baseline in the 24-hour ambulatory SBP was -16.6 mm Hg (95% CI, -18.8 to -14.3) in the baxdrostat group and -2.6 mm Hg (-4.7 to -0.4) in the placebo group. These results produced the estimated placebo-corrected difference of -14.0 mm Hg (-17.2 to -10.8; *P*<0.0001). Adverse events were reported among 52% of participants in the baxdrostat group and 37% in the placebo group, but only one serious adverse event occurred in each group.

The findings indicate that baxdrostat can significantly decrease 24-hour ambulatory SBP in individuals with resistant hypertension. The researchers concluded that, while steroidal and nonsteroidal mineralocorticoid receptor antagonists have displayed clinical benefits in certain populations with heart failure or CKD, selective aldosterone synthase inhibitors, such as baxdrostat, are a promising new treatment approach.

IGA NEPHROPATHY

Atrasentan as an Add-On in IgAN and Proteinuria

J Am Soc Nephrol. doi:10.1681/ASN.0000001076

People with IgA nephropathy (IgAN) and proteinuria have a risk of kidney failure even if they receive the guideline-recommended treatment of RAS inhibitors and sodium-glucose cotransporter-2 (SGLT2) inhibitors. Although atrasentan, a selective endothelin-A receptor antagonist, decreases proteinuria in IgA nephropathy (IgAN), its use as an adjunct to the other two medications has not undergone rigorous testing. To address this gap, **Hiddo J. L. Heerspink, PhD**, and colleagues investigated whether atrasentan would offer an additional benefit with an acceptable safety profile.

Researchers conducted a randomized, double-blind, placebo-controlled, crossover study involving participants with IgAN who were receiving stable, maximal doses of RAS inhibitors and SGLT2 inhibitors. They randomly assigned participants to receive 0.75 mg of atrasentan once per day during period 1, followed by a placebo during period 2 or vice versa, with the placebo and atrasentan in reverse order. The experiment allowed 12 weeks for washout between periods 1 and 2.

The primary end point was the difference in UPCR at 12 weeks, and the secondary end point was the difference in UPCR at 24 weeks. Safety end points included the incidence and severity of adverse events.

Of the 54 participants, the mean age was 48, 43% were female, the median UPCR was 1.0 g/g (interquartile range, 0.7-1.4), and the mean eGFR was 63 mL/min/1.73 m². Treatment with atrasentan versus placebo led to a difference in UPCR at week 12 of -25.3% (95% CI, -36.8 to -11.7; $P < 0.001$). The UPCR treatment difference between atrasentan versus placebo in period 2 at 24 weeks was -26.4% (95% CI, -45.8 to -0.0). Atrasentan was not discontinued during the trial due to treatment-related adverse events, and no deaths were recorded.

The findings suggest that atrasentan added to usual treatment can lead to a clinically meaningful decrease in proteinuria without safety concerns in this population, according to the authors.

Iptacopan Slows Kidney Function Decline in IgAN

N Engl J Med. doi:10.1056/NEJMoa2600743

Overactivation of the alternative complement pathway worsens IgAN and glomerular inflammation. In a phase 3 clinical trial, **Jonathan Barratt, PhD**, and colleagues showed that iptacopan, a complement factor B inhibitor, decreased 24-hour UPCR by 38.3% without causing safety concerns.

The analysis included adults with IgAN, a 24-hour UPCR of 1 gg or higher, and an eGFR of at least 30 mL/min/1.73 m² of body surface area despite supportive care. Researchers randomly assigned the participants 1:1 to receive 200 mg oral iptacopan or a placebo twice daily. They evaluated safety in addition to efficacy.

The primary end point was the annualized total eGFR slope, estimated over a 24-month period. Secondary end points included a composite kidney failure end point, including a sustained eGFR of less than 15 mL/min/1.73 m², a sustained

decline in eGFR of 30% or greater, initiation of maintenance dialysis, kidney transplant, or death from kidney failure.

Among 477 patients in the trial, 238 received iptacopan and 239 received placebo. In the iptacopan group, the annualized eGFR slope was -3.10 mL/min/1.73 m², and -6.12 mL/min/1.73 m² in the placebo group. The difference between the two groups was 3.02 mL/min/1.73 m² (95% CI, 2.02-4.01; adjusted $P < 0.001$).

Adverse events occurred in 87.0% of the iptacopan group and in 89.1% of the placebo group. Serious adverse events occurred in 12.2% and 11.7%, respectively. No deaths were recorded.

The results demonstrate that iptacopan significantly slows kidney function decline when compared with a placebo. Researchers considered the safety profile acceptable.

NEPHROLITHIASIS

Does Increased Hydration Prevent Urinary Tract Stones?

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Clinicians recommend increasing fluid intake to reduce the risk of recurrent urinary stones, but patient adherence is challenging. Citing a lack of research on the effectiveness of this intervention, **Alana C. Desai, MD**, and colleagues investigated whether a program promoting high fluid intake could reduce stone occurrence.

This clinical trial included participants with a history of urinary stones and low 24-hour urine volume who were enrolled at six US academic medical centers. Researchers randomly assigned participants 1:1 to a behavioral intervention to promote higher fluid intake or to a control group. The intervention involved a fluid prescription, monetary incentives to adhere to the fluid prescription, and coaching to address challenges in consuming more fluids.

The primary outcome was recurrence of urinary stones within a 2-year follow-up period. Secondary outcomes included urinary symptoms, change in 24-hour urine volume, growth of existing stone(s), and new stone formation.

Of the 1,658 participants assigned to intervention (n=826) and control (n=832) groups, 57% were female, and the median age was 44 years. At a median follow-up of 738 days, 19% of the intervention group and 20% of the control group experienced symptomatic stone events. While 24-hour urine volume rose in both groups, the intervention group had a greater increase at months 6, 12, 18, and 24. Symptoms of frequency, nocturia, and urgency occurred more often among the intervention group at months 6 and 12 but not at other times.

No statistical differences in symptomatic stone recurrence, new stone formation, or stone growth of at least 2 mm were found between groups.

The results suggest that the behavioral intervention did not reduce stone recurrence but increased urine volume. Future research should explore alternative adherence strategies or secondary prevention strategies beyond raising fluid intake. ●



Transitional Care Management

The revenue opportunity many nephrology practices are missing



Sarah Tolson

By Sarah Tolson

Hospitalizations remain one of the most vulnerable transition points for patients of nephrologists. Whether they are managing advanced CKD, end-stage renal disease (ESRD), hypertension, acute kidney injury, or dialysis-related complications, nephrologists routinely care for patients with medically complex conditions who have high rates of readmission. In many cases, nephrology professionals are already performing the clinical work associated with transitional care management (TCM) services after hospital discharge. However, many practices continue billing standard office-based evaluation and management (E/M) services instead of using the TCM codes specifically designed by CMS to recognize and reimburse this higher level of coordinated care.

The result is that nephrology practices may be missing both a meaningful reimbursement opportunity and a chance to standardize care coordination processes that improve patient outcomes.

Understanding TCM Services

Billed under *Current Procedural Terminology (CPT)* codes 99495 and 99496, TCM services support patients during the critical 30-day period after discharge from an inpatient or observational setting. For patients receiving kidney care, this period is often medically fragile and operationally complex. Medication regimens change; dialysis orders may be adjusted; laboratory monitoring is required; and communication among hospitals, dialysis facilities, primary care professionals, and specialists becomes essential.

Many nephrologists already perform this work instinctively. They review discharge summaries, reconcile medications, arrange follow-up visits, communicate with dialysis units, and answer patients' or caregivers' questions after discharge. The challenge is often not whether the work is being performed—it is—but whether the practice has implemented a standardized workflow that ensures all CMS requirements are consistently met and documented.

Under CMS guidelines, TCM requires several key elements:

- Interactive contact with the patient or caregiver within 2 business days of discharge
- Medication reconciliation and management
- Moderate or high complexity medical decision-making
- A face-to-face visit within 14 days for CPT code 99495 or within 7 days for CPT code 99496
- Ongoing non-face-to-face care coordination during the 30-day period after discharge

Importantly, CMS allows clinical staff operating under general supervision to perform many of the non-face-to-face components of care. Nurses, medical assistants, care coordinators, and advanced practice providers can all support compliant TCM workflows.

Financial and Clinical Opportunities

From a financial perspective, the opportunity is substantial. Many post-hospitalization nephrology follow-up visits are currently billed as established patient office visits such as CPT code 99214. However, when all TCM requirements are met, those encounters may instead qualify for CPT code 99495 or 99496 and are generally reimbursed at a significantly higher rate than standard office-based E/M services.

For many practices, the reimbursement difference between a CPT code 99214 and a TCM code may range from approximately \$200 to \$300 per encounter, depending on payer contracts and geographic adjustments. Across a nephrology population with frequent hospitalizations, the cumulative impact can become meaningful.

More importantly, TCM services recognize and reimburse work that nephrology practices are often already doing outside the exam room. Traditional office visit coding frequently fails to capture the extensive coordination, communication, medication management, and transition planning required for patients with complex kidney care needs after discharge.

The clinical benefits are equally important.

Patients with CKD and ESRD frequently experience fragmented care transitions. Hospital discharge instructions may conflict with dialysis treatment plans, medication changes may not be communicated clearly, and follow-up testing can be delayed. Missed dialysis treatments or confusion surrounding dry weight adjustments can quickly lead to readmissions.

A standardized TCM program creates a structured framework to address these gaps proactively. Early outreach allows practices to identify problems before they escalate. Medication reconciliation can uncover discrepancies between discharge medications and the patient's prior nephrology regimen. Prompt follow-up visits provide opportunities to reassess volume status, BP control, dialysis adequacy, and vascular access concerns.

Implementation May Be Easier Than You Realize

Fortunately, implementing TCM services does not necessarily require building an entirely new infrastructure. In many cases, success comes from formalizing processes that already exist.

Practices should develop workflows that identify discharged patients in real time and ensure timely patient outreach. Electronic health record templates can help capture required documentation elements, including discharge date, date of patient contact, medication reconciliation, medical decision-making complexity, and face-to-face visit timing.

Nephrology practices are uniquely positioned to succeed with TCM because coordinated chronic disease management is already central to nephrology care delivery. Dialysis rounds, multidisciplinary communication, and frequent patient touch-points are already embedded into most nephrology workflows.

Transitional care management offers nephrology practices an opportunity to improve continuity of care while strengthening financial sustainability.

TCM simply allows practices to structure, document, and appropriately reimburse the transitional care work they are already performing more intentionally.

As healthcare continues shifting toward value-based care and outcome-driven reimbursement models, TCM offers nephrology practices an opportunity to improve continuity of care while strengthening financial sustainability. For practices caring for patients with medically complex conditions and kidney disease requiring frequent hospitalizations, standardized TCM services represent not only a reimbursement opportunity but also a meaningful extension of high-quality, patient-centered nephrology care. ●

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